

## CONSUMPTION OF A TOXIC PLANT (*ZIGADENUS PANICULATUS*) BY MULE DEER

William S. Longland<sup>1,2</sup> and Charlie Clements<sup>1</sup>

*Key words:* death camas, *Zigadenus*, mule deer, poisonous plants, coevolution.

The abundance of green vegetation in nature can yield false impressions of the availability of food resources to herbivores because many plants have evolved anti-herbivore defenses. Defensive mechanisms commonly include incorporation of distasteful or toxic secondary chemical compounds into plant tissues. Effects of different compounds on consumers range from mild (unpalatable) to severe (illness or death from poisoning). Herbivores have consequently evolved a host of means for coping with defensive compounds, resulting in an evolutionary arms race between plants and herbivores (Freeland and Janzen 1974). Although evidence of plant/herbivore coevolution can be found for herbivores ranging from phytophagous insects to mega-vertebrates, we concentrate specifically on mule deer (*Odocoileus hemionus*) feeding on toxic plants.

Because domestic grazing animals lack a coevolutionary history with the plant communities in which they forage, they are often affected by toxic secondary compounds to a greater degree than native herbivores. This has significant economic impact on the range livestock industry due to direct losses, such as death, reduced fecundity, or reduced weight gain, and to indirect costs of minimizing such losses (Nielsen et al. 1988, James et al. 1992). Historical familiarity with local plant assemblages has provided herbivores foraging in their native ranges with two advantages over introduced domestic counterparts (Freeland and Janzen 1974, Laycock 1978, Laycock et al. 1988). First, native mammals often avoid eating toxic plant species that are eaten by domestic grazers. For example, toxic plants eaten by livestock, such as azalea (*Azalea* spp.) and larkspur (*Delphinium* spp.), are avoided by mule deer even when these plants are abundant

(Dixon 1934). Second, in most cases of native ungulates eating a plant species that is toxic to domestic animals, the plant does not produce noticeable toxic effects in the former, indicating that native herbivores may possess detoxification mechanisms for some plant toxins (Laycock 1978). Thus, deer consume without adverse effects a variety of plants poisonous to livestock (Stoddart and Rasmussen 1945, Dean and Winward 1974). Reciprocal examples in which native plants are toxic to native herbivores, but benign to domestic animals, are lacking in the literature.

Herein we report on four years of observations of an eastern Sierra Nevada mule deer herd feeding on substantial quantities of foothill death camas (*Zigadenus paniculatus*), a liliaceous bulb plant that is toxic to domestic sheep, cattle, and horses (Fleming et al. 1921, Kingsbury 1964, James et al. 1980, Panter et al. 1987). The genus *Zigadenus* includes several species, all containing toxic steroidal alkaloids (James et al. 1980). Death camas emerges earlier than most plants, making it particularly hazardous for spring grazing of livestock (Panter and James 1989). These plants have been variously described as “the most important poisonous plants in the western U.S.” (Kingsbury 1964) and “the most dangerous poisonous plants in North America” (Clarke and Clarke 1975). Foothill death camas has been described as one of the more toxic *Zigadenus* species (Kingsbury 1964, James et al. 1980).

Our study site is located at T20N, R18E, S36 just west of Reno, NV, on an alluvial fan at the southern base of Peavine Mountain. Woody vegetation is dominated by basin big sagebrush (*Artemisia tridentata tridentata*) and bitterbrush (*Purshia tridentata*). Death camas emerges at this site in mid-March, flowers in

<sup>1</sup>USDA, Agricultural Research Service, 920 Valley Road, Reno, NV 89512.

<sup>2</sup>Address correspondence to this author.

April, and remains green into May. A herd of mule deer, usually numbering 20–25 animals, has foraged extensively in this area from October to May since we began making observations in fall 1988.

We first noticed deer consuming death camas on 28 March 1989 (before plants flowered) and confirmed this with additional observations in all subsequent years. Examination of death camas foliage immediately after deer left the foraging patches consistently revealed fresh herbivore damage. We found that deer herbivory left a characteristic leaf damage pattern, with most or all leaves of a foraged plant cleanly bitten off perpendicular to their long axes. In addition to direct observations of deer consuming death camas, fresh deer pellet groups were found in patches of plants exhibiting this characteristic damage pattern during all five springs (1989–1993). During observation periods we found no evidence of deer exhibiting toxic effects from death camas consumption, and neither we nor personnel from the Nevada Department of Wildlife (which surveys deer in the area by air) have found any fresh deer carcasses in the vicinity.

Each year from 1990 through 1993 we walked 10–12 permanently located, parallel transects and categorized all death camas plants seen as either eaten or uneaten by deer. Transects were 500 m long, 20 m wide (i.e., we generally saw all plants occurring <10 m from the transect lines), and spaced 30 m apart. Usually, deer removed only the distal 2–5 cm of leaves, but on several occasions we found plants eaten to within 2 cm of ground level. Plants were considered eaten regardless of the amount of leaf removed. We tested these data for temporal differences in frequency of death camas consumption by comparing numbers of eaten versus uneaten plants among the four years of the study using a G-test of independence. We similarly tested for spatial effects on consumption by comparing eaten versus uneaten plant counts among individual transect lines within years.

There are at least two potential explanations for the partial consumption of leaves that we noted. Perhaps ends of leaves are less toxic than leaf bases, and deer preferentially consume less-toxic plant parts. Kingsbury (1964) suggests that death camas bulbs are the most toxic part of the plants, and a gradient of decreasing toxicity could occur from bulbs to

ends of leaves. Alternately, deer may occasionally sample plants in their environment (Free-land and Janzen 1974), and removal of short leaf segments may represent cautious sampling of a plant deer find undesirable. The latter possibility (sampling) seems less likely than the former (selectivity) because we have observed individual deer feeding on several death camas plants consecutively. Furthermore, total numbers of plants consumed on our transects were several orders of magnitude greater than the number of deer foraging in the study area, and it seems unlikely that deer would have to sample repeatedly so many plants to discover they are undesirable.

We found significant annual variation in the frequency of death camas consumption, ranging from 3.8% to 18.9% of total plants counted showing evidence of deer herbivory ( $G = 232.8$ ,  $df = 3$ ,  $P < .0001$ ; Table 1). Maximum and minimum percentages of plants eaten (Table 1) illustrate that frequency of herbivory also varied spatially; in each of the four years we sampled there was significant variation among transects in numbers of plants eaten ( $P < .001$  for all years). While the minority of plants in the local death camas population were eaten, the values in Table 1 also represent a surprisingly high frequency of herbivory on a plant species with such a notorious reputation.

The relatively low proportions of damaged plants indicate that deer may be selective for particular death camas plants. This is supported by the fact that deer generally ate only a few non-neighboring plants from large patches of death camas; rarely did the majority of plants within a patch show evidence of herbivory. The apparently selective use of individual death camas plants, significant temporal and spatial variation in death camas use, and infrequent extensive herbivory on small patches of plants could be due to variation among plants or patches in toxicity or to differing availabilities of superior foods leading to variation in the use of toxic foods.

Our observations suggest that death camas is more palatable to deer than to domestic cattle or sheep. Domestic animals must be force-fed death camas in captivity experiments (Fleming 1918, Fleming et al. 1921, Panter et al. 1987) and must be stressed or left with few alternative foods in nature before they consume it (Panter et al. 1987). Mule deer at our study site, however, occur at a low density and consume



TABLE 1. Numbers and percentages of foothill death camas plants consumed by mule deer along 500-m transects, 1990–1993, at Peavine Mountain (Washoe County, NV).

Year	Number of transects	Number of plants <sup>a</sup>		Plants eaten per transect (%)		
		Total	Eaten	Maximum	Minimum	$\bar{X} \pm \text{S.D.}$
1990	12	2646	501	29.0	7.3	18.6 $\pm$ 10.6
1991	12	2726	259	44.7	2.6	16.4 $\pm$ 13.4
1992	10	3073	118	32.6	1.6	8.3 $\pm$ 9.6
1993	10	3799	202	15.4	2.5	8.0 $\pm$ 4.0

<sup>a</sup>Includes combined data from all transects.

death camas each spring although alternative plants are available. Because bitterness is a general property of alkaloids (Laycock 1978), death camas is quite bitter. Most herbivores apparently find bitterness distasteful (Laycock 1978); however, bitterbrush (*Purshia tridentata*), which is named for its bitterness, is a preferred browse plant of mule deer. Although bitterbrush is also consumed by domestic ungulates, it is not highly preferred by them, perhaps because bitterness is a greater feeding deterrent to domestic animals than to deer.

Native herbivores have been observed consuming a variety of plant species known to be toxic to domestic herbivores (Laycock 1978), including an anecdotal report of mule deer in Utah consuming death camas and several other toxic plants (Stoddart and Rasmussen 1945). Recent work stimulates the interesting possibility that herbivores consume specific toxic plants to rid themselves of gut parasites (Barbosa et al. 1991, Gauld and Gaston 1992). However, this hypothesis only addresses why toxic plants are consumed rather than why the consumers are physiologically able to tolerate the toxins. Although we can only speculate about reasons mule deer are less affected by death camas toxicity than domestic ruminants, a likely explanation is that deer possess rumen microflora that have acquired the ability through natural selection to detoxify this plant (Freeland and Janzen 1974, Laycock 1978). Such selection is perhaps to be expected for native ruminants because the microflora community has seen prolonged exposure to native toxic plants. It is certainly possible, however, that deer are able to detoxify death camas by some other mechanism. For example, since deer are browsers, their diets include large amounts of tannins (Cooper and Owen-Smith 1985, Robbins et al. 1987) that may precipitate the alkaloids in death camas into a harmless tannate (Freeland and Janzen 1974).

Because even limited past exposure of a herbivore to a particular toxin can result in reduced toxic effects, selection for detoxifying rumen microflora may also account for intra-specific variation in toxicity among individuals of a domestic species. Such individual variation in susceptibility to death camas toxicity has been reported in force-feeding experiments with domestic sheep (Fleming et al. 1921, Kingsbury 1964). Perhaps it is possible to utilize this individual variation in selectively breeding for reduced vulnerability to particular toxins. Currently, most domestic grazing animals are products of artificial selection for productivity, rather than for resistance to environmental challenges.

Another avenue for applied research concerns the possibility of ameliorating effects of toxic plants through the transfer of rumen inocula from animals resistant to specific toxins to those that are susceptible. Jones (1985) reported that transfer of rumen cultures from goats that were resistant to poisoning by *Leucaena leucocephala* to susceptible goats and steers eliminated adverse effects of *Leucaena* consumption in the previously susceptible animals. This example suggests that even inter-specific transfer of rumen fluids may effectively reduce toxic effects in some cases.

Deer herbivory we witnessed on Peavine Mountain may affect the demography of the local death camas population. Defoliation experiments indicate that death camas probably suffers reduced reproductive output after herbivory (Tepedino 1982, Knapp 1986). While plants adapted to herbivory may compensate for loss of biomass by allocating additional energy to growth and/or reproduction, highly toxic species instead employ an evolutionary strategy of defense against herbivory and thus may not exhibit compensation (Cates 1975, Laycock 1978). When such defenses are circumvented by herbivores with detoxification

mechanisms, toxic plants should experience reduced fitness.

#### ACKNOWLEDGMENTS

We thank Dr. Jeanne Chambers, Dr. Kip Panter, and two anonymous reviewers for thoughtful reviews of the manuscript. This paper is a contribution of the USDA, Agricultural Research Service, Conservation Biology of Rangelands Unit, Reno, NV.

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Received 12 October 1993

Accepted 30 August 1994